

## HYPERTROPHIC CARDIOMYOPATHY

## Hypertrophic Obstructive Cardiomyopathy: Preoperative Echocardiographic Predictors of Outcome After Septal Myectomy

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**Objectives.** The purpose of this study was to determine whether two-dimensional and Doppler echocardiography are predictive of clinical outcome in patients with hypertrophic obstructive cardiomyopathy who undergo septal myectomy.

**Background.** Surgical myectomy provides excellent relief of symptoms in most patients with hypertrophic obstructive cardiomyopathy who are severely symptomatic despite medical therapy. There is a subset of patients who will remain symptomatic even after operation. Because comprehensive two-dimensional and Doppler echocardiography can define the range of anatomic and associated pathophysiologic abnormalities, it was hypothesized that preoperative echocardiographic variables may be predictive of clinical outcome after septal myectomy.

**Methods.** The clinical, electrocardiographic (ECG), echocardiographic and surgical data of 47 adult patients with hypertrophic cardiomyopathy who underwent isolated septal myectomy from 1986 to 1992 were analyzed. Specific symptoms were evaluated both preoperatively and at 1 year postoperatively. Electrocardiography and echocardiography were performed preopera-

tively and postoperatively. The ECG and echocardiographic variables were analyzed to determine whether any were predictive of residual symptoms 1 year postoperatively.

**Results.** The mean ( $\pm$ SD) age of the patients was  $47 \pm 15$  years. All were in New York Heart Association functional class III or IV. Dyspnea was present in all 47 patients and was severe in 70%. Most patients experienced symptomatic improvement at 1 year; dyspnea persisted in 26 patients (55%). The preoperative echocardiographic variables of asymmetric hypertrophy, severe systolic anterior motion of the mitral leaflet(s) and prolonged isovolumetric relaxation time were independent predictors of mild or no residual dyspnea postoperatively.

**Conclusions.** This initial study shows that the preoperative echocardiographic variables of asymmetric hypertrophy, severe systolic anterior motion of the mitral leaflet(s) and prolonged isovolumetric relaxation time can identify patients who are most likely to benefit from septal myectomy.

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Septal myectomy has become the treatment of choice for patients with hypertrophic obstructive cardiomyopathy and symptoms refractory to medical therapy. The purpose of surgical myectomy is to relieve the dynamic left ventricular outflow obstruction and decrease the coexistent mitral regurgitation (1). Although multiple studies have demonstrated dramatic relief of symptoms in most patients who undergo this procedure (1-6), a subset of patients continue to have residual symptoms even after the operation.

A wide spectrum of pathophysiologic processes contribute to the symptoms and signs of hypertrophic cardiomyopathy. These include dynamic left ventricular outflow obstruction, mitral regurgitation, diastolic dysfunction and arrhythmias (7,8). It is postulated that in some patients the predominant

abnormality is one of systole (outflow obstruction and mitral regurgitation) (8), whereas other patients have symptoms primarily due to diastolic dysfunction (9). The beneficial effect of surgical myectomy may be greatest in patients with predominantly systolic abnormalities. Noninvasive two-dimensional and Doppler echocardiography are able to identify the different anatomic and pathophysiologic abnormalities that occur in patients with hypertrophic obstructive cardiomyopathy. The purpose of this study was to determine whether preoperative two-dimensional and Doppler echocardiographic characteristics can identify patients who will benefit most from septal myectomy-induced relief of the systolic abnormalities.

### Methods

**Baseline data collection.** The clinical, electrocardiographic (ECG), echocardiographic and surgical data of 65 consecutive patients, 20 to 70 years old, with hypertrophic cardiomyopathy who underwent septal myectomy from 1986 to 1992 were reviewed retrospectively. The diagnosis of hypertrophic obstructive cardiomyopathy was confirmed by the characteristic echocardiographic findings of marked left ventricular hyper-

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trophy and rest or provokable left ventricular outflow obstruction.

Eighteen patients were excluded from this analysis to eliminate confounding factors contributing to postoperative outcome. Excluded were three patients who underwent emergency operation; six who underwent coronary artery bypass graft surgery or nonemergency mitral valve repair or replacement in addition to the septal myectomy; one in whom bilateral diaphragmatic paralysis developed postoperatively; one with significant aortic stenosis who underwent concomitant aortic valve replacement; and three with minimal or no symptoms preoperatively (one patient was asymptomatic but had a very strong family history of sudden cardiac death and a high rest left ventricular outflow pressure gradient). Three patients died in the early postoperative period. One patient was lost to follow-up. The remaining 47 patients formed the study group who underwent isolated septal myectomy for New York Heart Association functional class III and IV symptoms. Three of these 47 patients did have minor aortic valve procedures at the time of the septal myectomy: 1 had a repair procedure after sustaining a retraction injury, and 2 had decalcification procedures of calcified but nonstenotic valves.

Specific symptoms, such as dyspnea, angina, near syncope and syncope, were evaluated as part of the preoperative clinical assessment of patients, as was overall functional class. Dyspnea or angina was considered mild if it occurred only with strenuous activity, moderate if it occurred with normal daily activities and severe if it occurred with minimal activity or at rest.

**Electrocardiographic measurements.** The ECG was recorded at preoperative clinical evaluation. Left atrial enlargement was present if the negative terminal deflection of the P wave in lead  $V_1$  was  $\geq 40$  ms in duration (10). Q waves were considered abnormal if  $>40$  ms in duration and  $\geq 25\%$  of the R wave in depth. Giant negative T waves were defined as T wave inversion  $\geq 10$  mV. Left ventricular hypertrophy was defined by the criteria of Sokolow and Lyon (11) ( $SV_1 + RV_5$  or  $RV_6 > 35$  mV). The postoperative ECG was recorded shortly before hospital dismissal.

**Echocardiographic measurements.** Comprehensive two-dimensional and Doppler echocardiographic studies (12,13) were performed in all 47 patients preoperatively and in 46 postoperatively before hospital dismissal. These studies were all recorded on 0.75-in. videotape, and subsequent off-line analysis was performed without knowledge of clinical outcome. Two-dimensional and Doppler measurements were made with a Dextra digitizing computer.

The pattern of hypertrophy was determined; predominantly septal hypertrophy was classified as asymmetric. In all patients with asymmetric hypertrophy, septal wall thickness was at least 1.3 times that of the posterior wall. In all patients with diffuse (concentric) hypertrophy, the difference between septal and posterior wall thickness was  $\leq 2$  mm.

Left ventricular hypertrophy was quantitated with the hypertrophy point score (7) and by calculation of the left ventricular mass index. Mean left ventricular wall thickness

was calculated by measuring the wall thickness of each of 16 segments (14) obtained from short-axis two-dimensional images of the left ventricle at the basal, midventricular and apical levels and then averaging them. An estimate of the left ventricular mass index was then calculated through use of the mean wall thickness in a corrected cube function formula (15). Left atrial volume was measured with the biplane area-length method (16). Systolic anterior motion of the mitral valve leaflet(s) was graded mild if the mitral leaflet did not come within 10 mm of the septum during systole; moderate if the mitral leaflet came within 10 mm of the septum or established brief septal contact; and severe if there was prolonged mitral leaflet-septal contact (7,17,18).

Doppler variables of systolic and diastolic function were measured from the recorded spectral display of continuous wave left ventricular outflow and pulsed wave mitral inflow velocity curves. Left ventricular ejection time was measured from the left ventricular outflow velocity curve and corrected for gender and heart rate using established regression equations (19). Peak left ventricular outflow pressure gradient at baseline (rest) and after provocation (with the Valsalva maneuver or amyl nitrite inhalation) was calculated by applying the simplified Bernoulli equation to the peak systolic velocity of the continuous wave Doppler signal across the left ventricular outflow tract (20). Diastolic filling variables were recorded from a pulsed wave sample volume placed at the tip of the mitral leaflets, as previously described (13). Measurements included the early (E wave) and late (A wave) mitral inflow velocities, E wave acceleration and deceleration times, A wave duration and isovolumetric relaxation time. Intraventricular flow during the isovolumetric relaxation period was identified and measured with use of continuous wave Doppler from the apical window. Tricuspid regurgitation velocity was measured with continuous wave Doppler. Color flow Doppler was used for qualitative assessment of the severity of mitral regurgitation (none, grades 1/4, 2/4, 3/4, 4/4).

**Surgical technique.** The presence of left ventricular outflow obstruction was considered to be the qualifying criterion for surgical myectomy, irrespective of the morphology of the left ventricle. Standard cardiopulmonary bypass techniques were used with moderate ( $25^\circ$  to  $32^\circ\text{C}$ ) systemic hypothermia. Myocardial protection was achieved with cold blood or crystalloid potassium cardioplegia with topical cooling. The subaortic obstruction was approached by way of an aortotomy, and the septal resection was begun by making two parallel, longitudinal incisions, the first beneath the nadir of the right coronary cusp and the second beneath the commissure between the right and left coronary cusps. The incisions were joined superiorly, and a bar of muscle was excised past the bulging septum to the level of the papillary muscles. Additional myocardium was removed between this resection and the lateral attachment of the anterior mitral leaflet. For the patient with midventricular obstruction, further resection was done below the level of the papillary muscles. The left ventricular outflow tract was then inspected and palpated for complete-

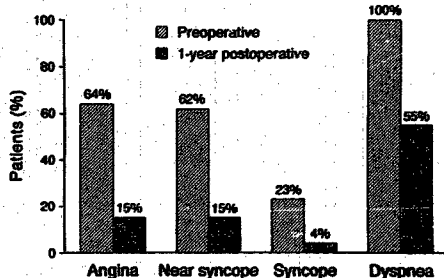


Figure 1. Symptoms in 47 study patients. New syncope developed in two patients who were syncope free preoperatively.

ness of relief of outflow obstruction, and the aortotomy was repaired.

**Clinical outcome.** Follow-up information at the end of the first postoperative year (mean  $\pm$  SD)  $12.2 \pm 3.8$  months) was obtained from return clinic visits, mailed questionnaires, correspondence from referring physicians or telephone calls to the patients. Included in the follow-up assessment was a detailed description of the presence and severity of persistent symptoms, such as dyspnea, angina, near syncope and syncope.

**Statistical analysis.** Data were summarized, both overall and by group, by mean value  $\pm$  SD or by frequency percents. Groups were compared with the Wilcoxon rank-sum test or Pearson chi-square test. Preoperative to postoperative changes in echocardiographic factors were tested with the Wilcoxon signed-rank test. Univariate and multivariate associations between preoperative ECG and echocardiographic variables and preoperative and 1-year postoperative symptoms were assessed by ordinal logistic regression. Because of the large number of potential predictor variables, the exploratory nature of these analyses is recognized, although no formal adjustment for multiple comparisons was attempted because of the adverse effect on power. Instead, the empirical results were considered for their mechanistic plausibility.

## Results

**Clinical presentation.** Dyspnea was the most common symptom and was present in all 47 patients (Fig. 1); 33 (70%) had severe dyspnea (Fig. 2). Other symptoms were angina in 30 patients (64%), near syncope in 29 (62%) and syncope in 11 (23%). All patients were in functional class III or IV.

**Medications.** Patients had been receiving various regimens of cardiac drugs, some of which could not be tolerated. At the time of preoperative assessment, 44 patients (94%) were taking a beta-adrenergic or calcium channel blocking agent, and 13 (28%) were receiving both drugs. Beta-blockers were used in 35 patients (74%) and calcium channel blockers in 22 (47%). Five patients (11%) were taking disopyramide, three (6%) digoxin and one amiodarone.

**Preoperative ECG.** Six patients (13%) were in atrial fibrillation, and the remaining 41 were in sinus rhythm. Of those in

## Dyspnea

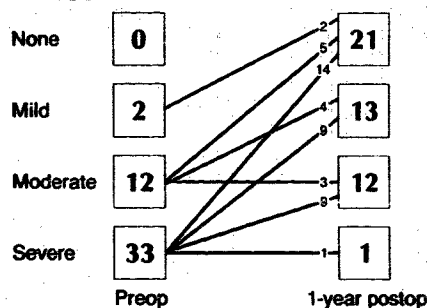


Figure 2. Preoperative (Preop) and postoperative (postop) severity of dyspnea in 47 study patients. Postoperatively, 26 patients (55%) had at least mild dyspnea, and 13 (28%) had at least moderate dyspnea.

sinus rhythm, 30 (73%) had left atrial enlargement, 10 (24%) had a PR interval  $>200$  ms, and 10 (24%) had a PR interval = 200 ms. The shortest PR interval was 140 ms and the longest 320 ms. Left bundle branch block was present in three patients (6%). In those without left bundle branch block, 4 (9%) had pathologic Q waves, 2 (5%) had giant inverted T waves, and 25 (57%) had left ventricular hypertrophy.

**Preoperative two-dimensional echocardiography.** The pattern of left ventricular hypertrophy was variable: 6 (13%) of the 47 patients had diffuse (concentric) hypertrophy; 1 had predominantly midventricular hypertrophy; and remaining 40 (85%) had asymmetric septal hypertrophy with or without anterolateral extension of hypertrophy. During systole, midventricular obliteration was seen in nine patients (19%) and apical obliteration in five (11%). Maximal left ventricular wall thickness averaged  $25.1 \pm 4.3$  mm (range 18 to 33), and mean wall thickness was  $17.6 \pm 1.9$  mm. Mean left ventricular mass index was  $165 \pm 51$  g/m<sup>2</sup> (normal range 110 to 130), and mean left atrial volume was  $84 \pm 40$  ml (normal 50  $\pm$  15). Systolic anterior motion of the mitral leaflet(s) was severe in 36 patients (77%), moderate in 9 (19%) and mild in 2 (4%).

**Preoperative Doppler echocardiography.** The average peak rest left ventricular outflow pressure gradient was  $64 \pm 38$  mm Hg (range 0 to 180). Peak provoked gradient, determined in 27 patients, averaged  $87 \pm 46$  mm Hg (range 10 to 196). Six patients had a peak rest gradient  $<25$  mm Hg, five of whom had provoked gradients  $<50$  mm Hg. Significant gradients were provoked at the time of cardiac catheterization in these five patients. The average corrected left ventricular ejection time was prolonged at  $432 \pm 27$  ms (range 382 to 483; normal corrected ejection time  $411 \pm 13$ ) (19). Isovolumetric relaxation time averaged  $100 \pm 24$  ms (range 67 to 168); 22 patients (50%) had a prolonged isovolumetric relaxation time ( $\geq 100$  ms). Mitral inflow Doppler signal measurements were variable: E/A ratio  $1.4 \pm 0.6$  (range 0.5 to 2.7), E wave peak velocity  $1.0 \pm 0.3$  m/s (range 0.6 to 1.6), A wave peak velocity  $0.8 \pm 0.3$  m/s (range 0.3 to 1.6) and E wave deceleration time  $232 \pm 73$  ms (range 88 to 440). By color Doppler jet area

assessment, mitral regurgitation was at least moderate (2/4) in 27 patients (58%), mild in 15 (32%) and trivial or absent in 5 (10%). Intraventricular isovolumetric relaxation flow directed toward the apex was identified in 21 (70%) of 30 patients and varied from 0.5 to 1.5 m/s (mean 0.9).

**Postoperative two-dimensional/Doppler echocardiography.** A paired comparison of the preoperative and postoperative echocardiographic variables was performed in patients in whom both were measured. The peak rest left ventricular outflow gradient decreased to  $23 \pm 20$  mm Hg postoperatively ( $p = 0.0001$  vs. preoperative gradient). Corrected left ventricular ejection time shortened to  $387 \pm 26$  ms ( $p = 0.0001$ ). Systolic anterior motion of the mitral leaflet(s) remained severe in 7 (15%) patients (vs. 35 [76%] preoperatively) and was moderate in 10 (22%), mild in 14 (30%) and absent in 15 (33%) ( $p = 0.0001$ ). Only 8 patients (17%) had moderate (2/4) or greater mitral regurgitation (vs. 26 [57%] preoperatively), and 21 (46%) had no or trivial mitral regurgitation ( $p = 0.0001$ ). Overall, there was no significant postoperative change in the isovolumetric relaxation time, E wave deceleration time, A wave peak velocity, E/A ratio and left atrial volume. E wave peak velocity decreased to  $0.9 \pm 0.3$  m/s ( $p < 0.01$ ). The six patients with diffuse (concentric) hypertrophy tended to have a lower postoperative peak rest left ventricular outflow gradient ( $10 \pm 5$  vs.  $25 \pm 21$  mm Hg,  $p = 0.07$ ) and had a similar low frequency of residual severe systolic anterior motion (0% vs. 18%,  $p = \text{NS}$ ) and mitral regurgitation  $\geq 2/4$  (0% vs. 20%,  $p = \text{NS}$ ) compared with patients with asymmetric hypertrophy.

**Postoperative clinical outcome.** The 1-year postoperative evaluation revealed fewer and less severe symptoms (Fig. 1). Forty patients (85%) had no angina; 40 (85%) had no near syncope; and all 11 patients with preoperative syncope had no postoperative syncope. New syncope developed postoperatively in two patients. Twenty-six patients (55%) had residual dyspnea (Fig. 2), and 13 patients (28%) had moderate or severe residual dyspnea. Postoperatively, these patients had a peak rest left ventricular outflow gradient similar to those with no or mild residual dyspnea ( $19 \pm 18$  vs.  $24 \pm 21$  mm Hg,  $p = \text{NS}$ ) and a similar frequency of residual mitral regurgitation  $\geq 2/4$  (8% vs. 21%,  $p = \text{NS}$ ) and severe systolic anterior motion (8% vs. 18%,  $p = \text{NS}$ ).

**Preoperative echocardiographic predictors of clinical outcome.** Twenty-six echocardiographic (Table 1) and three ECG variables were analyzed vis-à-vis preoperative and postoperative symptoms at 1 year. The ECG variables tested were left ventricular hypertrophy, left atrial enlargement and rhythm (sinus rhythm vs. atrial fibrillation). No echocardiographic or ECG variable was predictive of preoperative symptoms or postoperative angina or near syncope. By multivariate analysis, the echocardiographic variables of asymmetric hypertrophy, severe systolic anterior motion of the mitral leaflet(s) and prolonged isovolumetric relaxation time emerged as independent predictors of a lesser degree of residual dyspnea (Table 2). Patients with two or more of these favorable echocardiographic predictor variables were unlikely to have significant postoperative dyspnea (Fig. 3).

**Table 1. Two-Dimensional and Doppler Echocardiographic Variables Measured**

Two-dimensional
Hypertrophy pattern
Hypertrophy point score (Wigle)
LV maximal wall thickness
LV mean wall thickness
LV mass
LV mass index
LV end-diastolic dimension
LV end-systolic dimension
Fractional shortening
Ejection fraction
SAM severity (Henry)
Left atrial dimension
Left atrial volume
Doppler
Corrected LV ejection time
Peak rest LVO gradient
Peak provoked LVO gradient
IVRT
Isovolumetric relaxation flow
E wave peak velocity
A wave peak velocity
E/A ratio
E wave acceleration time
E wave deceleration time
A wave duration
MR severity
TR peak velocity

IVRT = isovolumetric relaxation time; LV = left ventricular; LVO = left ventricular outflow; MR = mitral regurgitation; SAM = systolic anterior motion (of mitral leaflet); TR = tricuspid regurgitation.

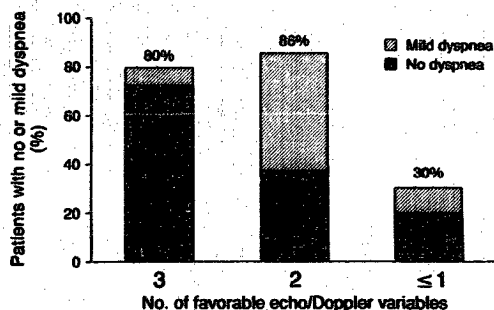
## Discussion

Most patients who undergo surgical treatment of hypertrophic obstructive cardiomyopathy show substantial clinical improvement. The present study reaffirms that septal myectomy is an efficacious procedure in patients who are incapacitated despite optimal medical treatment. In particular, patients with angina or syncope enjoy a near complete resolution of symptoms. Patients with dyspnea also improve, although not to the same extent. In some patients, as shown, in the present study, although the septal myectomy procedure is technically successful, with subsequent decrease in left ventricular outflow gradient, severity of mitral valve systolic anterior motion and mitral regurgitation, improvement in dyspnea may be minimal. This lack of change is because dyspnea is related in part to

**Table 2. Multivariate Predictors of Mild or No Residual Dyspnea One Year Postoperatively**

Variable	OR (95% CI)	p Value
Asymmetric hypertrophy	7.85 (1.3-45.5)	0.022
Severe SAM	4.71 (1.2-18.6)	0.027
Prolonged IVRT	4.40 (1.3-15.4)	0.019

CI = confidence interval; OR = odds ratio; other abbreviations as in Table 1.



**Figure 3.** Postoperative dyspnea status of 14 patients with three, 23 with two and 10 with or one or no favorable preoperative echocardiographic (echo)/Doppler variables (asymmetric hypertrophy, severe systolic anterior motion of mitral leaflet(s) [prolonged mitral leaflet-septal contact] and prolonged isovolumetric relaxation time  $\geq 100$  ms)]. The subgroup with two favorable variables includes three patients in whom isovolumetric relaxation time could not be measured.

abnormal diastolic function, which may not be improved by septal myectomy.

**Two-dimensional predictors of outcome.** To our knowledge, the present study is the first to attempt to predict who may or may not benefit from surgical intervention on the basis of preoperative echocardiographic data. An asymmetric pattern of left ventricular hypertrophy as opposed to a diffuse (concentric) pattern on preoperative echocardiography was one of three independent predictors of symptomatic improvement. Severe hypertrophy produces increased chamber stiffness and decreased muscle compliance, a major contributor to diastolic dysfunction. This would not be expected to change after septal myectomy. In addition, the increased muscle mass adversely affects left ventricular relaxation by nonuniformity and abnormal contraction coupling, neither of which would be affected by relief of outflow obstruction. Thus, the pattern of diffuse (concentric) hypertrophy may be a preoperative marker of severe diastolic dysfunction that cannot be improved with surgical myectomy.

The severity of the systolic anterior motion of the mitral valve was also a predictor of symptomatic improvement after septal myectomy. The degree of mitral valve systolic anterior motion was previously used as an indirect marker of the severity of the outflow obstruction. However, the degree of systolic anterior motion of the mitral leaflet(s) may provide additional information, such as the degree of original displacement of the mitral valve apparatus and the amount of distortion of the mitral valve leaflet(s) during systole caused by the Venturi effect. These factors relate not only to the degree of obstruction but also to the concomitant mitral regurgitation that ensues. In addition, the amount of tissue removed at operation is relatively small, and thus it is not only the physical enlargement of the outflow tract, but also the interruption of the pathophysiologic sequence of events that is the mechanism of the efficacy of septal myectomy. Thus, patients with the severest systolic anterior motion of the mitral leaflet(s) would

be expected to benefit most from septal myectomy. The left ventricular outflow gradient itself was not a statistical predictor in this study.

**Doppler predictors of outcome.** The finding of a prolonged isovolumetric relaxation time predicting symptomatic relief was an interesting result of the present study. Isovolumetric relaxation time is an indirect measure of the rate of left ventricular relaxation, and patients with a prolonged isovolumetric relaxation time may be those with a slowed rate of relaxation. Although ventricular relaxation is complex, it is related to the contraction load of the left ventricle (7). Thus, patients with the longest isovolumetric relaxation time who respond symptomatically to operation may be those in whom a high contraction load can be relieved at myectomy. Alternatively, the patients with a prolonged isovolumetric relaxation time may be those who have lower left atrial pressure, because mitral valve opening is delayed in patients with lower left ventricular filling pressures.

None of the other diastolic function variables were predictive of symptomatic outcome in the present study. Mitral flow velocity curves have been used in previous studies of other disease states as measures of diastolic filling of the left ventricle. However, these mitral flow velocities have not been predictive of symptoms or prognosis in previous studies of patients with hypertrophic cardiomyopathy (21,22). These flow curves are influenced by multiple factors, including patient age, severity of mitral regurgitation, ventricular and atrial compliance, left ventricular relaxation and left atrial pressure (23). These factors vary widely in patients with hypertrophic cardiomyopathy, and simple mitral flow velocities may therefore not be indicative of the degree of diastolic dysfunction or predictive of postoperative outcome.

**Study limitations.** Limitations of the present study include its retrospective nature. A selection bias cannot be entirely excluded; clinicians may not have referred certain patients for septal myectomy if they thought that there was predominantly severe diastolic dysfunction. There was limited cardiac catheterization data, and absolute left-sided pressures were therefore not available for analysis. The Doppler mitral inflow signal was analyzed without knowledge of the pulmonary vein velocity curves, which were not sampled routinely in the late 1980s. These curves may have provided additional information with regard to the presence of "pseudonormal" mitral inflow signals, which are seen as the left atrial pressure increases (24). The power of the statistical analysis was modest, given the multiple variables tested and the size of the study group. Prospective evaluation of these echocardiographic/Doppler predictors of clinical outcome would therefore be desirable. Preoperative and postoperative symptoms such as dyspnea are, to some extent, dependent on the perceptions of both patient and physician. More objective measures of postoperative exercise capacity, such as exercise time on a treadmill or maximal oxygen-carrying capacity, were not obtained for analysis.

**Conclusions.** Most patients with hypertrophic obstructive cardiomyopathy will benefit symptomatically from surgical myectomy; however, a subset will continue to have significant

residual dyspnea after operation. The present initial study has shown that the presence of the preoperative echocardiographic variables of asymmetric hypertrophy, severe systolic anterior motion of the mitral leaflet(s) and prolonged isovolumetric relaxation time can identify patients who will have no or mild residual dyspnea postoperatively and thus be most likely to benefit from septal myectomy.

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